

\$%^STN;HighlightOn= \*\*\*;HighlightOff=\*\*\* ;

Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSPTAHXK1654

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

\*\*\*\*\* Welcome to STN International \*\*\*\*\*

- NEWS 1 Web Page URLs for STN Seminar Schedule - N. America
- NEWS 2 "Ask CAS" for self-help around the clock
- NEWS 3 FEB 27 New STN AnaVist pricing effective March 1, 2006
- NEWS 4 APR 04 STN AnaVist \$500 visualization usage credit offered
- NEWS 5 MAY 10 CA/CAPLUS enhanced with 1900-1906 U.S. patent records
- NEWS 6 MAY 11 KOREAPAT updates resume
- NEWS 7 MAY 19 Derwent World Patents Index to be reloaded and enhanced
- NEWS 8 MAY 30 IPC 8 Rolled-up Core codes added to CA/CAPLUS and USPTAFULL/USPAT2
- NEWS 9 MAY 30 The F-Term thesaurus is now available in CA/CAPLUS
- NEWS 10 JUN 02 The first reclassification of IPC codes now complete in INPADOC
- NEWS 11 JUN 26 TULSA/TULSA2 reloaded and enhanced with new search and display fields
- NEWS 12 JUN 28 Price changes in full-text patent databases EPFULL and PCTFULL
- NEWS 13 JUL 11 CHEMSAFE reloaded and enhanced
- NEWS 14 JUL 14 FSTA enhanced with Japanese patents
- NEWS 15 JUL 19 Coverage of Research Disclosure reinstated in DWPI
- NEWS EXPRESS JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.
- NEWS HOURS STN Operating Hours Plus Help Desk Availability
- NEWS LOGIN Welcome Banner and News Items
- NEWS IPC8 For general information regarding STN implementation of IPC 8
- NEWS X25 X.25 communication option no longer available

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

\*\*\*\*\* STN Columbus \*\*\*\*\*

FILE 'HOME' ENTERED AT 15:13:55 ON 21 JUL 2006

|                      |            |         |
|----------------------|------------|---------|
| => file caplus       |            |         |
| COST IN U.S. DOLLARS | SINCE FILE | TOTAL   |
|                      | ENTRY      | SESSION |
| FULL ESTIMATED COST  | 0.21       | 0.21    |

FILE 'CAPLUS' ENTERED AT 15:14:19 ON 21 JUL 2006  
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.  
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.  
COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

Copyright of the articles to which records in this database refer is held by the publishers listed in the PUBLISHER (PB) field (available for records published or updated in Chemical Abstracts after December

26, 1996), unless otherwise indicated in the original publications.  
The CA Lexicon is the copyrighted intellectual property of the  
American Chemical Society and is provided to assist you in searching  
databases on STN. Any dissemination, distribution, copying, or storing  
of this information, without the prior written consent of CAS, is  
strictly prohibited.

FILE COVERS 1907 - 21 Jul 2006 VOL 145 ISS 5  
FILE LAST UPDATED: 20 Jul 2006 (20060720/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply.  
They are available for your review at:

<http://www.cas.org/infopolicy.html>

=> e reduced glutathione/cn  
\*\*\* REGISTRY INITIATED \*\*\*  
Substance data EXPAND from CAS REGISTRY in progress...

|     |       |                                                                    |
|-----|-------|--------------------------------------------------------------------|
| E1  | 1     | REDUCED FMN/CN                                                     |
| E2  | 1     | REDUCED FOLATE CARRIER PROTEIN (HUMAN GENE RFC EXON 1 FRAGMENT)/CN |
| E3  | 1 --> | REDUCED GLUTATHIONE/CN                                             |
| E4  | 1     | REDUCED GLUTATHIONE LITHIUM SALT/CN                                |
| E5  | 1     | REDUCED GLUTATHIONE PEROXIDASE/CN                                  |
| E6  | 1     | REDUCED GLUTATHIONE SODIUM SALT/CN                                 |
| E7  | 1     | REDUCED GLUTATHIONE THIOLATE ANION/CN                              |
| E8  | 1     | REDUCED GREEN FFB/CN                                               |
| E9  | 1     | REDUCED HALOPERIDOL OXIDASE/CN                                     |
| E10 | 1     | REDUCED HBS/CN                                                     |
| E11 | 1     | REDUCED HEMATIN/CN                                                 |
| E12 | 1     | REDUCED HOPKINSIAXANTHIN/CN                                        |

=> s e3  
\*\*\* REGISTRY INITIATED \*\*\*  
Substance data SEARCH and crossover from CAS REGISTRY in progress...  
Use DISPLAY HITSTR (or FHITSTR) to directly view retrieved structures.

L2 41636 L1

=> d hitrn

L2 ANSWER 1 OF 41636 CAPLUS COPYRIGHT 2006 ACS on STN  
IT \*\*\*70-18-8\*\*\* , Reduced glutathione, biological studies  
RL: BSU (Biological study, unclassified); BIOL (Biological study)  
(oxygenation-ozonation induced biochem. modifications in blood)

=> s 70-18-8/THU and dillon j?/au  
L3 1 70-18-8/THU AND DILLON J?/AU

=> d

L3 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN  
AN 2004:999661 CAPLUS <<LOGINID::20060721>>  
DN 141:388779  
TI Methods and compositions for protecting against cataract development  
associated with vitrectomies  
IN \*\*\*Dillon, James\*\*\*  
PA USA  
SO U.S. Pat. Appl. Publ., 41 pp.  
CODEN: USXXCO  
DT Patent

LA English

FAN.CNT 1

|      | PATENT NO.                                                                                                                                                                                                                                                                                                                                                                                        | KIND | DATE     | APPLICATION NO. | DATE     |
|------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------|----------|-----------------|----------|
| PI   | US 2004229814                                                                                                                                                                                                                                                                                                                                                                                     | A1   | 20041118 | US 2003-650357  | 20030827 |
|      | WO 2005048920                                                                                                                                                                                                                                                                                                                                                                                     | A2   | 20050602 | WO 2004-US27700 | 20040825 |
|      | W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW |      |          |                 |          |
|      | RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG                                                                                                                    |      |          |                 |          |
| PRAI | US 2002-406907P                                                                                                                                                                                                                                                                                                                                                                                   | P    | 20020828 |                 |          |
|      | US 2003-650357                                                                                                                                                                                                                                                                                                                                                                                    | A    | 20030827 |                 |          |

=> s us20040229814/pn

L4 1 US20040229814/PN

=> select l4 1 rn

E1 THROUGH E4 ASSIGNED

=> file reg

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

11.65

18.42

FILE 'REGISTRY' ENTERED AT 15:17:38 ON 21 JUL 2006

USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.

PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

COPYRIGHT (C) 2006 American Chemical Society (ACS)

Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 20 JUL 2006 HIGHEST RN 894992-91-7

DICTIONARY FILE UPDATES: 20 JUL 2006 HIGHEST RN 894992-91-7

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

Please note that search-term pricing does apply when conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

<http://www.cas.org/ONLINE/UG/regprops.html>

=> s e1-e4

L5 4 (70-18-8/BI OR 50-81-7/BI OR 7727-37-9/BI OR 7782-44-7/BI)

=> s 50-81-7/rn

L6 1 50-81-7/RN

=> d

L6 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2006 ACS on STN

RN \*\*\*50-81-7\*\*\* REGISTRY

ED Entered STN: 16 Nov 1984

CN L-Ascorbic acid (8CI, 9CI) (CA INDEX NAME)

OTHER NAMES:

CN (+)-Ascorbic acid

CN 3-keto-L-Gulofuranolactone

CN 3-Oxo-L-gulofuranolactone  
 CN Adenex  
 CN Allercorb  
 CN Antiscorbic vitamin  
 CN Antiscorbutic vitamin  
 CN Ascoltin  
 CN Ascorbajen  
 CN Ascorbic acid  
 CN Ascorbicap  
 CN Ascorbutina  
 CN Ascorell  
 CN Ascorin  
 CN Ascorteal  
 CN Ascorvit  
 CN C-L 6/PW  
 CN C-Quin  
 CN C-Vimin  
 CN Cantan  
 CN Cantaxin  
 CN Catavin C  
 CN Ce-Mi-Lin  
 CN Ce-Vi-Sol  
 CN Cebicure  
 CN Cebion  
 CN Cebione  
 CN Cecon  
 CN Cegiolan  
 CN Ceglion  
 CN Ceklin  
 CN Celaskon  
 CN Celin  
 CN Cell C  
 CN Cemagyl  
 CN Cenetone  
 CN Cereon  
 CN Cergona  
 CN Cescorbat  
 CN Cetamid  
 CN Cetane  
 CN Cetane-Caps TC  
 CN Cetebe  
 CN Cetemican  
 CN Cevalin  
 CN Cevatine  
 CN Cevex  
 CN Cevimin  
 CN Cevital  
 CN Cevitamic acid

ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for  
DISPLAY

FS STEREOSEARCH

DR 884381-69-5, 623158-95-2, 56533-05-2, 57304-74-2, 57606-40-3, 56172-55-5,  
 129940-97-2, 14536-17-5, 50976-75-5, 154170-90-8, 89924-69-6, 30208-61-8,  
 259133-78-3

MF C6 H8 O6

CI COM

LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN\*, BIOSIS,  
 BIOTECHNO, CA, CABA, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS,  
 CHEMINFORMRX, CHEMLIST, CIN, CSCHM, CSNB, DDFU, DETHERM\*, DRUGU,  
 EMBASE, ENCOMPLIT, ENCOMPLIT2, ENCOMPPAT, ENCOMPPAT2, GMELIN\*, HSDB\*,  
 IFICDB, IFIPAT, IFIUDB, IMSCOSEARCH, IPA, MEDLINE, MRCK\*, MSDS-OHS,  
 NAPRALERT, PHAR, PIRA, PROMT, PS, RTECS\*, SPECINFO, SYNTHLINE,  
 TOXCENTER, TULSA, ULIDAT, USAN, USPAT2, USPATFULL, VETU, VTB  
 (\*File contains numerically searchable property data)

Other Sources: DSL\*\*, EINECS\*\*, TSCA\*\*, WHO

(\*\*Enter CHEMLIST File for up-to-date regulatory information)

Absolute stereochemistry.

/ Structure 1 in file .gra /

\*\*PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT\*\*

78764 REFERENCES IN FILE CA (1907 TO DATE)  
1729 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA  
78897 REFERENCES IN FILE CAPLUS (1907 TO DATE)  
12 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> s 7727-37-9/rn  
L7 1 7727-37-9/RN

=> d

L7 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2006 ACS on STN  
RN \*\*\*7727-37-9\*\*\* REGISTRY  
ED Entered STN: 16 Nov 1984  
CN Nitrogen (8CI, 9CI) (CA INDEX NAME)  
OTHER NAMES:  
CN Diatomic nitrogen  
CN Dinitrogen  
CN Molecular nitrogen  
CN Nitrogen (N2)  
CN Nitrogen gas  
CN Nitrogen nutrition (plant)  
CN Nitrogen-14  
FS 3D CONCORD  
DR 778548-56-4, 745765-07-5, 794449-54-0, 161728-27-4, 156457-45-3,  
93037-13-9, 263005-65-8  
MF N2  
CI COM  
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOSIS, BIOTECHNO, CA, CABA,  
CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN,  
CSCHEM, CSNB, DDFU, DETHERM\*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2,  
ENCOMPPAT, ENCOMPPAT2, GMELIN\*, HSDB\*, IFICDB, IFIPAT, IFIUDB, IPA,  
MEDLINE, MRCK\*, MSDS-OHS, PIRA, PROMT, RTECS\*, SPECINFO, TOXCENTER,  
TULSA, ULIDAT, USAN, USPAT2, USPATFULL, VTB  
(\*File contains numerically searchable property data)  
Other Sources: DSL\*\*, EINECS\*\*, TSCA\*\*  
(\*\*Enter CHEMLIST File for up-to-date regulatory information)

/ Structure 2 in file .gra /

\*\*PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT\*\*

296486 REFERENCES IN FILE CA (1907 TO DATE)  
12797 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA  
296781 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> s 7782-44-7/rn  
L8 1 7782-44-7/RN

=> d

L8 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2006 ACS on STN  
RN \*\*\*7782-44-7\*\*\* REGISTRY  
ED Entered STN: 16 Nov 1984  
CN Oxygen (8CI, 9CI) (CA INDEX NAME)  
OTHER NAMES:  
CN Dioxygen  
CN Molecular oxygen  
CN Oxygen molecule  
FS 3D CONCORD  
DR 1338-93-8, 14797-70-7, 80217-98-7, 80937-33-3  
MF O2  
CI COM  
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOSIS, BIOTECHNO, CA, CABA,  
CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMINFORMRX, CHEMLIST, CHEMSAFE, CIN,  
CSCHEM, CSNB, DDFU, DETHERM\*, DRUGU, EMBASE, ENCOMPLIT, ENCOMPLIT2,  
ENCOMPPAT, ENCOMPPAT2, GMELIN\*, HSDB\*, IFICDB, IFIPAT, IFIUDB, IPA,

MEDLINE, MRCK\*, MSDS-OHS, PIRA, PROMT, PS, RTECS\*, SPECINFO, TOXCENTER,  
TULSA, ULIDAT, USAN, USPAT2, USPATFULL, VTB  
(\*File contains numerically searchable property data)  
Other Sources: DSL\*\*, EINECS\*\*, TSCA\*\*  
(\*\*Enter CHEMLIST File for up-to-date regulatory information)

/ Structure 3 in file .gra /

\*\*PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT\*\*

385963 REFERENCES IN FILE CA (1907 TO DATE)  
35594 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA  
386372 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> d his

(FILE 'HOME' ENTERED AT 15:13:55 ON 21 JUL 2006)

FILE 'CAPLUS' ENTERED AT 15:14:19 ON 21 JUL 2006

FILE 'REGISTRY' ENTERED AT 15:14:41 ON 21 JUL 2006  
E REDUCED GLUTATHIONE/CN

FILE 'CAPLUS' ENTERED AT 15:14:41 ON 21 JUL 2006  
S E3

L1 FILE 'REGISTRY' ENTERED AT 15:14:47 ON 21 JUL 2006  
1 S E3/CN

L2 FILE 'CAPLUS' ENTERED AT 15:14:48 ON 21 JUL 2006  
41636 S L1  
L3 1 S 70-18-8/THU AND DILLON J?/AU  
L4 1 S US20040229814/PN  
SELECT L4 1 RN

FILE 'REGISTRY' ENTERED AT 15:17:38 ON 21 JUL 2006  
L5 4 S E1-E4  
L6 1 S 50-81-7/RN  
L7 1 S 7727-37-9/RN  
L8 1 S 7782-44-7/RN

=> s (vitreous(A)replacement) and 70-18-8 and 50-81-7  
L9 0 (VITREOUS(A)REPLACEMENT) AND 70-18-8 AND 50-81-7

=> s (vitreous(A)replacement) and 70-18-8/rn  
L10 0 (VITREOUS(A)REPLACEMENT) AND 70-18-8/RN

=> e vitreous replacement/ct  
'CT' IS NOT A VALID EXPAND FIELD CODE FOR FILE 'REGISTRY'  
The indicated field code is not available for EXPAND in this  
file. To see a list of valid EXPAND field codes, enter HELP  
SFIELDS at an arrow prompt (=>).

=> file caplus

| COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION |
|----------------------|------------------|---------------|
| FULL ESTIMATED COST  | 32.22            | 50.64         |

FILE 'CAPLUS' ENTERED AT 15:27:46 ON 21 JUL 2006  
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.  
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.  
COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

Copyright of the articles to which records in this database refer is held by the publishers listed in the PUBLISHER (PB) field (available for records published or updated in Chemical Abstracts after December 26, 1996), unless otherwise indicated in the original publications. The CA Lexicon is the copyrighted intellectual property of the

American Chemical Society and is provided to assist you in searching databases on STN. Any dissemination, distribution, copying, or storing of this information, without the prior written consent of CAS, is strictly prohibited.

FILE COVERS 1907 - 21 Jul 2006 VOL 145 ISS 5  
FILE LAST UPDATED: 20 Jul 2006 (20060720/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply. They are available for your review at:

<http://www.cas.org/infopolicy.html>

=> e vitreous replacement/ct

| E#  | FREQUENCY | AT  | TERM                                |
|-----|-----------|-----|-------------------------------------|
| E1  | 0         | 2   | VITREOUS HUMOR BARRIER BLOOD/CT     |
| E2  | 1337      | 94  | VITREOUS MATERIALS/CT               |
| E3  | 0         | --> | VITREOUS REPLACEMENT/CT             |
| E4  | 0         | 2   | VITREOUS SEMICONDUCTORS/CT          |
| E5  | 0         | 16  | VITREOUS SILICA/CT                  |
| E6  | 0         | 2   | VITREOUS SILICA SYNTHETIC FIBERS/CT |
| E7  | 127       | 5   | VITREOUS STATE/CT                   |
| E8  | 0         | 2   | VITREOUS STRUCTURE/CT               |
| E9  | 0         | 2   | VITREOUS SUBSTANCES/CT              |
| E10 | 0         | 1   | VITREUM/CT                          |
| E11 | 0         | 1   | VITREUS/CT                          |
| E12 | 0         | 1   | VITRIC/CT                           |

=> s cataract/obi

L11 6087 CATARACT/OBI

=> s 70-18-8 and 50-81-7

\*\*\* REGISTRY INITIATED \*\*\*

Substance data SEARCH and crossover from CAS REGISTRY in progress...  
Use DISPLAY HITSTR (or FHITSTR) to directly view retrieved structures.

L13 78897 L12

\*\*\* REGISTRY INITIATED \*\*\*

Substance data SEARCH and crossover from CAS REGISTRY in progress...  
Use DISPLAY HITSTR (or FHITSTR) to directly view retrieved structures.

L15 41636 L14

L16 4494 L15 AND L13

=> d his

(FILE 'HOME' ENTERED AT 15:13:55 ON 21 JUL 2006)

FILE 'CAPLUS' ENTERED AT 15:14:19 ON 21 JUL 2006

FILE 'REGISTRY' ENTERED AT 15:14:41 ON 21 JUL 2006  
E REDUCED GLUTATHIONE/CN

FILE 'CAPLUS' ENTERED AT 15:14:41 ON 21 JUL 2006  
S E3

FILE 'REGISTRY' ENTERED AT 15:14:47 ON 21 JUL 2006  
1 S E3/CN

FILE 'CAPLUS' ENTERED AT 15:14:48 ON 21 JUL 2006  
41636 S L1  
1 S 70-18-8/THU AND DILLON J?/AU  
1 S US20040229814/PN  
SELECT L4 1 RN

FILE 'REGISTRY' ENTERED AT 15:17:38 ON 21 JUL 2006  
4 S E1-E4  
1 S 50-81-7/RN  
1 S 7727-37-9/RN  
1 S 7782-44-7/RN  
0 S (VITREOUS(A)REPLACEMENT) AND 70-18-8 AND 50-81-7  
0 S (VITREOUS(A)REPLACEMENT) AND 70-18-8/RN

FILE 'CAPLUS' ENTERED AT 15:27:46 ON 21 JUL 2006  
E VITREOUS REPLACEMENT/CT  
6087 S CATARACT/OBI  
S 70-18-8/REG# AND 50-81-7/REG#

FILE 'REGISTRY' ENTERED AT 15:29:30 ON 21 JUL 2006  
1 S 50-81-7/RN

FILE 'CAPLUS' ENTERED AT 15:29:31 ON 21 JUL 2006  
78897 S L12

FILE 'REGISTRY' ENTERED AT 15:29:31 ON 21 JUL 2006  
1 S 70-18-8/RN

FILE 'CAPLUS' ENTERED AT 15:29:32 ON 21 JUL 2006  
41636 S L14  
4494 S L15 AND L13

=> s l16 and l11

L17 77 L16 AND L11

=> d ti

L17 ANSWER 1 OF 77 CAPLUS COPYRIGHT 2006 ACS on STN  
TI Formulation for preventing and treating \*\*\*cataract\*\*\* , degeneration  
of macula and other eye disease and its use

=> d ti

L17 ANSWER 1 OF 77 CAPLUS COPYRIGHT 2006 ACS on STN  
TI Formulation for preventing and treating \*\*\*cataract\*\*\* , degeneration  
of macula and other eye disease and its use

=> d ti 1-5

L17 ANSWER 1 OF 77 CAPLUS COPYRIGHT 2006 ACS on STN  
TI Formulation for preventing and treating \*\*\*cataract\*\*\* , degeneration  
of macula and other eye disease and its use

L17 ANSWER 2 OF 77 CAPLUS COPYRIGHT 2006 ACS on STN  
TI Methods and compositions for protecting against \*\*\*cataract\*\*\*  
development associated with vitrectomies

L17 ANSWER 3 OF 77 CAPLUS COPYRIGHT 2006 ACS on STN  
TI Protective effect of ascorbate against oxidative stress in the mouse lens

L17 ANSWER 4 OF 77 CAPLUS COPYRIGHT 2006 ACS on STN  
TI Dietary caloric restriction may delay the development of \*\*\*cataract\*\*\*  
by attenuating the oxidative stress in the lenses of Brown Norway rats



L17 ANSWER 5 OF 77 CAPLUS COPYRIGHT 2006 ACS on STN  
TI \*\*\*Cataract\*\*\* formation in Atlantic salmon, Salmo salar L., smolt  
relative to dietary pro- and antioxidants and lipid level

=> d his

(FILE 'HOME' ENTERED AT 15:13:55 ON 21 JUL 2006)  
FILE 'CAPLUS' ENTERED AT 15:14:19 ON 21 JUL 2006  
FILE 'REGISTRY' ENTERED AT 15:14:41 ON 21 JUL 2006  
E REDUCED GLUTATHIONE/CN  
FILE 'CAPLUS' ENTERED AT 15:14:41 ON 21 JUL 2006  
S E3  
FILE 'REGISTRY' ENTERED AT 15:14:47 ON 21 JUL 2006  
L1 1 S E3/CN  
FILE 'CAPLUS' ENTERED AT 15:14:48 ON 21 JUL 2006  
L2 41636 S L1  
L3 1 S 70-18-8/THU AND DILLON J?/AU  
L4 1 S US20040229814/PN  
SELECT L4 1 RN  
FILE 'REGISTRY' ENTERED AT 15:17:38 ON 21 JUL 2006  
L5 4 S E1-E4  
L6 1 S 50-81-7/RN  
L7 1 S 7727-37-9/RN  
L8 1 S 7782-44-7/RN  
L9 0 S (VITREOUS(A)REPLACEMENT) AND 70-18-8 AND 50-81-7  
L10 0 S (VITREOUS(A)REPLACEMENT) AND 70-18-8/RN  
FILE 'CAPLUS' ENTERED AT 15:27:46 ON 21 JUL 2006  
E VITREOUS REPLACEMENT/CT  
L11 6087 S CATARACT/OBI  
S 70-18-8/REG# AND 50-81-7/REG#  
FILE 'REGISTRY' ENTERED AT 15:29:30 ON 21 JUL 2006  
L12 1 S 50-81-7/RN  
FILE 'CAPLUS' ENTERED AT 15:29:31 ON 21 JUL 2006  
L13 78897 S L12  
FILE 'REGISTRY' ENTERED AT 15:29:31 ON 21 JUL 2006  
L14 1 S 70-18-8/RN  
FILE 'CAPLUS' ENTERED AT 15:29:32 ON 21 JUL 2006  
L15 41636 S L14  
L16 4494 S L15 AND L13  
L17 77 S L16 AND L11

=> s l17 not dillon j?/au  
L18 75 L17 NOT DILLON J?/AU

=> s l18 not py>2002  
L19 64 L18 NOT PY>2002

=> s l19 and vit?  
L20 36 L19 AND VIT?

=> d l20 1-15 ibib abs

L20 ANSWER 1 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 2003:286284 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 139:333032  
TITLE: Protective effect of Co-SZ eye drop on galactose  
\*\*\*cataract\*\*\* in rats  
AUTHOR(S): Qi, Mingxin; Huang, Xiurong; Wang, Zhaoyang; Wang,  
Yong; Zheng, Liangpu; Lin, Jiumao; Lin, Wei; Ye,  
Hongzhi

CORPORATE SOURCE: Department of Ophthalmology, the Second Affiliated Hospital, Fujian College of Traditional Chinese Medicine, Fuzhou, 350003, Peop. Rep. China  
SOURCE: Zhongguo Bingli Shengli Zazhi (2002), 18(10), 1206-1208  
CODEN: ZBSZEB; ISSN: 1000-4718  
PUBLISHER: Jinan Daxue  
DOCUMENT TYPE: Journal  
LANGUAGE: Chinese  
AB The effects of Co-SZ eye drop on galactose cataract were studied in rats. Based on folk remedy, SZ eye drop was made from leech, as a modified SZ eye drop, Co-SZ eye drop was enriched in Zinc and \*\*\*Vitamin\*\*\* C. The animal model of galactose cataract in SD rats was used. All animals were randomly divided into 3 groups: control group(using 0.9% NaCl instead of SZ and Co SZ), SZ group and Co-SZ group. Lens opacities were examd. dynamically in each groups via FS-3V slit-lamp microscope. Superoxide dismutase(SOD), glutathione peroxidase(GSH-Px), glutathione(GSH) and sol. protein(SP) in the lenses were measured in 15 days. Both the Co-SZ and SZ eye drops could significantly delay and alleviate galactose cataract in rats, with better effect of Co-SZ than SZ eye drop. The antioxidant index indicated that SOD, GSH-Px, GSH in Co-SZ and SZ group were significantly higher than that in control group. Furthermore, SOD, GSH-Px in Co-SZ group were higher than that in SZ group significantly. Co-SZ eye drops could significantly delay and alleviate galactose cataract in rats, the effect is much better than SZ eye drops. The different effect between SZ and Co-SZ eye drops could be raised from the different content of Zinc, which is involved in anti-oxidn.

L20 ANSWER 2 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 2002:516601 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 137:83653  
TITLE: Methods and compositions for treating  
\*\*\*cataracts\*\*\* using substances derived from yeast or saltbush with or without chromium  
INVENTOR(S): Mirsky, Nitsa  
PATENT ASSIGNEE(S): Natural Compounds Ltd., Israel  
SOURCE: U.S., 25 pp., Cont.-in-part of U.S. 395,534.  
CODEN: USXXAM  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 2  
PATENT INFORMATION:

| PATENT NO. | KIND | DATE     | APPLICATION NO. | DATE        |
|------------|------|----------|-----------------|-------------|
| US 6416794 | B1   | 20020709 | US 2000-617865  | 20000717    |
| US 6261606 | B1   | 20010717 | US 1999-395534  | 19990914    |
|            |      |          | US 1999-395534  | A2 19990914 |

PRIORITY APPLN. INFO.:  
AB Compns. and methods having anticataract and antiretinopathy activity comprise compds. extd. from natural resources including yeast and saltbush (Atriplex halimus) or synthetic chromium complexes. The compn. is administered orally, parenterally, topically or s.c. For example, the active fractions - GTF, isolated from yeast, and ACMS, isolated from saltbush - inhibited the activity of eye lens aldose reductase, an enzyme which plays an important role in the etiol. of diabetic cataract, by reducing the rate of NADPH oxidn.  
REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 3 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 2002:405161 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 137:121656  
TITLE: Studies on Singlet Oxygen Formation and UVA Light-mediated Photobleaching of the Yellow Chromophores in Human Lenses  
AUTHOR(S): Ortwerth, Beryl J.; Chemoganskiy, Vitaliy; Olesen, P. R.  
CORPORATE SOURCE: Mason Eye Institute, University of Missouri, Columbia, MO, 65212, USA  
SOURCE: Experimental Eye Research (2002), 74(2), 217-229  
CODEN: EXERA6; ISSN: 0014-4835  
PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE: Journal  
LANGUAGE: English

AB The protein-bound chromophores, which increase with aging in the human lens, act as UVA sensitizers, producing almost exclusively singlet oxygen in \*\*\*vitro\*\*\*. Direct irradiation of whole, aged human lenses with high intensity UVA light (200 mW cm<sup>-2</sup> for 24 h), however, failed to produce singlet oxygen damage, as evidenced by the lack of either His or Trp photodestruction. Total homogenates of human lenses prepared in a cuvette under air did show destruction of His and Trp residues by UVA light, but no destruction was seen when equivalent homogenates were prepared under argon. These data are consistent with the idea that the low oxygen levels in the lens prevent singlet oxygen damage in vivo. UVA irradiation of aged human lenses in culture caused an extensive photobleaching of the yellow chromophores. A time course indicated that the photobleaching increased with time, with significant color loss apparent after 6 h. Homogenization of the irradiated and dark control lenses in 6 M guanidine-HCl, followed by detection of the difference spectrum, showed approximately 50% bleaching of compounds with a  $\lambda_{\text{max}}$  at 355 nm. Similarly, fluorophores with a  $\lambda_{\text{max}}$  for excitation of 355 nm and for emission of 420 nm were 50% destroyed by the UVA light. Similar results were obtained in \*\*\*vitro\*\*\* by the anaerobic irradiation of a sonication-solubilized WI fraction from type II brunescent cataracts and from aged human lenses. In this system, there was an initial bleaching of 15% after 30 min of irradiation, followed by a slow increase over the next 6 h to a final bleaching of 30%. The addition of 1.0 mM ascorbic acid, but not 1.0 mM glutathione (GSH), increased the photobleaching to 60% under argon, and the loss of ascorbate could be detected under these anaerobic conditions. In the presence of air, UVA light produced no photobleaching, but rather caused a three-fold increase in absorbance at 345 nm, which was prevented by the inclusion of 1.0 mM ascorbic acid and almost 50% inhibited by 1.0 mM GSH. The data are consistent with the conversion of the triplet state of the sensitizers to anion and cation radicals in the absence of oxygen. Photobleaching may occur either by dismutation of the anion radical or by reduction of the anion radical by ascorbate via type I chemistry. UVA irradiation of an enriched fraction of sensitizers from a proteolytic digest from type II cataract lenses produced a 63% bleaching at 330 nm in the absence of oxygen, and the almost complete loss of the A330 absorbing and 350/450 nm fluorescent peaks upon HPLC separation. This loss correlated with the loss of the ability of the irradiated fraction to produce singlet oxygen in \*\*\*vitro\*\*\* upon subsequent UVA irradiation.

REFERENCE COUNT: 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 4 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:534722 CAPLUS <<LOGINID::20060721>>

DOCUMENT NUMBER: 136:288996

TITLE: Schisandrin B protects against oxidative damage of lens in \*\*\*vitro\*\*\*

AUTHOR(S): Huang, Xiurong; Qi, Mingxin; Ye, Hongzhi; Lin, Wei; Zheng, Liangpu; Lin, Jiumao

CORPORATE SOURCE: Fujian College of Traditional Chinese Medicine, Fuzhou, 350003, People's Republic of China

SOURCE: Zhongguo Yaoxue Zazhi (Beijing, China) (2001), 36(5), 310-313

CODEN: ZYZAEU; ISSN: 1001-2494

PUBLISHER: Zhongguo Yaoxue Zazhishe

DOCUMENT TYPE: Journal

LANGUAGE: Chinese

AB The protective effect of schisandrin B on experimental oxidative damage to lens was studied. Twenty New Zealand rabbits (40 eyes) were divided into four groups: control group, Fenton group (Fenton), pirenixine sodium group (PS), and 0.5 mM schisandrin B group (Sch B). All fresh lenses except control group were bathed in Fenton reaction system composed of H<sub>2</sub>O<sub>2</sub> and FeCl<sub>3</sub> as a model of oxidative damage of lens, and treated with PS or Sch B in CO<sub>2</sub> incubator for 24 h. The soluble protein (SP), superoxide dismutase (SOD), glutathione peroxidase (GSH-Px), glutathione (GSH), \*\*\*vitamin\*\*\* C (\*\*\*Vit\*\*\* C), and malondialdehyde (MDA) in homogenized lenses were measured. SP of lens in Sch B group was significantly higher than that in Fenton reaction system ( $P < 0.01$ ), and activities of SOD and GSH-Px and levels of GSH and \*\*\*Vit\*\*\* C in lens of Sch B group were increased and MDA decreased as compared with the Fenton group. SOD activity, GSH, and \*\*\*Vit\*\*\* C in Sch B group were 1.66, 2.58, and 2.36 times those

of PS group, resp., but MDA in Sch B group was 24% lower than that in PS group (P <0.01). The results showed that Sch B may remarkably protect lens against oxidative damage in Fenton reaction system, the anti-oxidative effect of Sch B was better than that of PS, and Sch B may be used as a potential drug to prevent and treat cataract.

L20 ANSWER 5 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:191254 CAPLUS <<LOGINID::20060721>>

DOCUMENT NUMBER: 135:221217

TITLE: Experimental study on natural antioxidants protecting lens against oxidative injuries

AUTHOR(S): Huang, Xiurong; Qi, Mingxin; Ye, Hongzhi; Lin, Wei; Zheng, Liangpu; Lin, Jiumao

CORPORATE SOURCE: Fujian College of Traditional Chinese Medicine, Fuzhou, 350003, Peop. Rep. China

SOURCE: Zhongguo Bingli Shengli Zazhi (2001), 17(2), 120-123  
CODEN: ZBSZEB; ISSN: 1000-4718

PUBLISHER: Jinan Daxue

DOCUMENT TYPE: Journal

LANGUAGE: Chinese

AB The protective effect of five natural antioxidants (schisandrin B, Sch B; silibinin, SIB; Pr gallate, PG; sodium ferulate, SF; total flavonoids of hippophase, TFH) on exptl. oxidative injuries of lens was studied. All fresh transparent lenses of rabbit eyes except control group were bathed in Fenton reaction system to produce a model of oxidative damages of lens, meanwhile Sch B, SIB, PG, SF, TFH and pirenoxine sodium (PS) were added in the reaction system in different groups resp. Lenses were incubated for 24 h. Then total protein (TP), sol. protein (SP), superoxide dismutase (SOD), glutathione peroxidase (GSH-Px), glutathione, \*\*\*vitamin\*\*\* C (\*\*\*Vit\*\*\* C), total activities of anti-oxidn. (TAO) and malondialdehyde (MDA) in homogenized lenses were measured to observe the effects of five antioxidants on above index. Five antioxidants increased the anti-oxidative index and decreased MDA in lenses of oxidative damages in different levels, the effects are better than that of PS, esp. in group SF and Sch B. The five natural antioxidants protected lens against exptl. oxidative injuries very well. There are wide prospects to pursue effective anti-cataract drugs from natural antioxidants.

L20 ANSWER 6 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:182802 CAPLUS <<LOGINID::20060721>>

DOCUMENT NUMBER: 135:135325

TITLE: Free radicals and antioxidants in ophthalmology

AUTHOR(S): Racek, J.; Holecek, V.; Ricarova, R.

CORPORATE SOURCE: Ostav klinicke biochemie a laboratorni diagnostiky, Lekarska Fakulta Univerzity Karlovy fakultni nemocnice, Plzen, Czech.

SOURCE: Klinicka Biochemie a Metabolismus (2001), 9(1), 20-24  
CODEN: KBMEFQ; ISSN: 1210-7921

PUBLISHER: Ceska Lekarska Spolecnost J. Ev. Purkyne

DOCUMENT TYPE: Journal; General Review

LANGUAGE: Czech

AB A review, with 30 refs. Oxygen, various chem. substances and UV rays exert a direct action on the eye and can cause the formation of free radicals in the eye. Therefore the antioxidant content of the eye is high. In \*\*\*vitreous\*\*\* fluid and the \*\*\*vitreous\*\*\* body it is in particular ascorbic acid, the lens contains a considerable amt. of reduced glutathione; the activity of antioxidant enzymes in different structures of the eye is also high. The most frequent and most serious eye diseases involving of free radicals are in particular cataract, retinopathies of different origin and mol. degeneration. The authors describe in the submitted review the possible mechanism of the development of these diseases with regard to metabolic processes generating free radicals and the part played by antioxidants in the protection of the eye and prevention of its damage by free radicals.

L20 ANSWER 7 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:52845 CAPLUS <<LOGINID::20060721>>

DOCUMENT NUMBER: 135:87118

TITLE: Inhibition of experimental oxidative damages of lens by sodium ferulate

AUTHOR(S): Huang, Xiu-Rong; Qi, Ming-Xin; Ye, Hong-Zhi; Lin, Wei; Zheng, Liang-Pu; Lin, Jiu-Mao

CORPORATE SOURCE: Central Laboratory, Fujian College of Traditional Chinese Medicine, Fuzhou, 350003, Peop. Rep. China

SOURCE: Zhongguo Yaolixue Yu Dulixue Zazhi (2000), 14(6), 430-433

CODEN: ZYYZEW; ISSN: 1000-3002

PUBLISHER: Zhongguo Yaolixue Yu Dulixue Zazhi Biarjibu

DOCUMENT TYPE: Journal

LANGUAGE: Chinese

AB To investigate the inhibition of exptl. oxidative damages of lens by sodium ferulate, 20 New Zealand rabbits (40 eyes) were divided randomly into four groups: control group, Fenton group (Fenton), pirenoxine sodium group (PS), and sodium ferulate group (SF). Eyeballs were extd. under the condition of sterility immediately. Lenses were drawn, bathed in different media of above groups and incubated in CO2 incubator for 24 h. Sol. protein (SP), superoxide dismutase (SOD), glutathione peroxidase (GSH-Px), glutathione (GSH), \*\*\*vitamin\*\*\* C (\*\*\*Vit\*\*\* C) and malondialdehyde (MDA) in homogenized lenses were measured. The results showed that SP, SOD, GSH-Px, GSH, \*\*\*Vit\*\*\* C of SF group were higher than those in Fenton and PS groups; MDA of SF group was lower than that in Fenton and PS groups. The results indicate that SF inhibits exptl. oxidative damages of lens, and it is more effective than PS. The study provides a scientific basis to prevent and treat cataract by using SF as a drug.

L20 ANSWER 8 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2000:568528 CAPLUS <<LOGINID::20060721>>

DOCUMENT NUMBER: 133:168395

TITLE: Orally ingested compositions for prevention and treatment of age-related eye disorders

INVENTOR(S): Gorsek, Wayne F.

PATENT ASSIGNEE(S): Vitacost Inc., USA

SOURCE: U.S., 3 pp.

CODEN: USXXAM

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

| PATENT NO.             | KIND | DATE     | APPLICATION NO. | DATE     |
|------------------------|------|----------|-----------------|----------|
| US 6103756             | A    | 20000815 | US 1999-372055  | 19990811 |
| PRIORITY APPLN. INFO.: |      |          | US 1999-372055  | 19990811 |

AB Disclosed is an oral compn. for prevention, stabilization, reversal and treatment of age-related macular degeneration, cataracts, elevated ocular pressure, diabetic retinopathy and glaucoma. One claimed compn. comprises \*\*\*vitamin\*\*\* C 100-600 mg, \*\*\*vitamin\*\*\* E 100-2000 IU, \*\*\*vitamin\*\*\* A 100-2000 IU, taurine 100-1000 mg, selenium 50-600 .mu.g, Bilberry ext. 40-1000 mg, lutein 6-100 mg, lycopene 6-100 mg, .alpha.-lipoic acid 50-1000 mg, quercetin 10-1000 mg, rutin 10-1000 mg, and citrus bioflavonoids 10-1000 mg.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 9 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2000:180661 CAPLUS <<LOGINID::20060721>>

DOCUMENT NUMBER: 133:72363

TITLE: Relationship between oxidative stress and galactose-induced \*\*\*cataract\*\*\*

AUTHOR(S): Han, Xiuxian; Chen, Jimin; Wang, Xiang; Chen, Zhuji; Deng, Xinguo; Ding, Xingzhen; Pang, Yingrin; Tian, Xiaoli; Zhou, Quan; Jin, Weimin; Li, Jianxin

CORPORATE SOURCE: Henan Inst. Ophthalmology, Zhengzhou, 450003, Peop. Rep. China

SOURCE: Yanke Yanjiu (1999), 17(1), 34-37

CODEN: YAYAFH; ISSN: 1003-0808

PUBLISHER: Henansheng Yanke Yanjiuso

DOCUMENT TYPE: Journal

LANGUAGE: Chinese

AB Objective To det. whether oxidative stress damage may be involved in galactose-induced cataract in vivo and in \*\*\*vitro\*\*\*. Methods A model of galactose-induced cataract in SD rat was established by injecting 50% galactose( 15 g/kg/d). An in \*\*\*vitro\*\*\* model was established by

incubation of lenses in an oxygen free radical generation system. The levels of MDA, SOD, GSH-px, CAT, GSH, \*\*\*Vitamin\*\*\* E and C were examd. Results We obsd. the morphol. and pathol. of lens opacity over time. The changes developed as follows: precystic-vesicle, cystic-vesicle, fusion cystic-vesicles and cortical stage. In lenses of the animal model, the MDA was significantly increased 2.5 fold, but GSH, SOD, and water sol. protein were decreased by 30%, 59% and 20%, resp. In incubated lenses, the MDA was also significantly increased 3.6 fold, but GSH, SOD GSH-PX, CAT, \*\*\*Vitamin\*\*\* E and C were decreased by 80%, 25%, 60%, 61%, 78% and 61%, resp. Conclusion It is important to det. the pathogenesis of galactose-induced cataract, osmotic damage and oxidative stress appear to be involved.

L20 ANSWER 10 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1999:558221 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 131:335320  
TITLE: Modelling cortical cataractogenesis. 21: In diabetic rat lenses taurine supplementation partially reduces damage resulting from osmotic compensation leading to osmolyte loss and antioxidant depletion  
AUTHOR(S): Mitton, K. P.; Linklater, H. A.; Dzialoszynski, T.; Sanford, S. E.; Starkey, K.; Trevithick, J. R.  
CORPORATE SOURCE: Department of Biochemistry, Faculty of Medicine and Dentistry, University of Western Ontario, London, ON, N6A 5C1, Can.  
SOURCE: Experimental Eye Research (1999), 69(3), 279-289  
CODEN: EXERA6; ISSN: 0014-4835  
PUBLISHER: Academic Press  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB The concn. of taurine and the amino acids, glutathione, cysteine, ascorbate and ATP were detd. in the lenses of rats made diabetic with streptozotocin. In the clear lenses, prior to vacuole formation after 1 or 2 wk of diabetes, the increase in concn. of sorbitol and the total decrease of all these osmolytes were not significantly different. The major components of the osmolytes lost were taurine and amino acids, which together accounted for over 75% of the total osmolyte loss. Since glutathione, ascorbate, taurine and cysteine have been reported to have antioxidant activity, it appears that their loss may potentiate damage occurring as a result of free radicals generated by nonenzymic glycation by the Maillard reaction. Amino acids also lost as a result of the osmotic compensation, are estd. to be responsible for almost half of the antioxidant activity lost. To test this hypothesis, normal and streptozotocin diabetic female Wistar rats were given taurine at 0.05% or 0.10% (wt./wt.) in the diet. This treatment resulted in small only marginally significant increases in serum taurine levels. At the end of 6 wk the rats were examd. for wt. gain or loss and at the time of killing, blood was collected for measurement of serum glucose. .gamma.-Crystallin levels were detd. in \*\*\*vitreous\*\*\* and aq. humors using a RIA. A lens from each rat was homogenized in 8 M guanidinium chloride for ATP anal. In normal rats, a small amt. of .gamma.-crystallin was found in the \*\*\*vitreous\*\*\* humor, and an even smaller amt. in the aq. humor. Diabetes caused a 4- to 5-fold increase in the \*\*\*vitreous\*\*\* humor and a 4-fold increase in .gamma.-crystallin in the aq. humor. Diabetes also led to a significant worsening in general body condition, loss of body wt., formation of cataracts, and decrease in lens ATP levels. Addn. of taurine to the diet of diabetic animals resulted in a significant decrease of .gamma.-crystallin leakage into the \*\*\*vitreous\*\*\* but not the aq. humor. Taurine had no effect on the lens ATP levels. Neither streptozotocin diabetes nor taurine in the diet appeared to affect the wt. of the lenses. (c) 1999 Academic Press.

REFERENCE COUNT: 69 THERE ARE 69 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 11 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1999:52834 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 130:208307  
TITLE: Contributions of polyol pathway to oxidative stress in diabetic \*\*\*cataract\*\*\*  
AUTHOR(S): Lee, Alan Y. W.; Chung, Stephen S. M.  
CORPORATE SOURCE: Institute of Molecular Biology, University of Hong Kong, Hong Kong, Peop. Rep. China

SOURCE: FASEB Journal (1999), 13(1), 23-30  
CODEN: FAJOEC; ISSN: 0892-6638  
PUBLISHER: Federation of American Societies for Experimental  
Biology  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB There is strong evidence to show that diabetes is assocd. with increased oxidative stress. The source of this oxidative stress remains unclear. Using transgenic mice that overexpress aldose reductase (AR) in their lenses, the authors found that the flux of glucose through the polyol pathway is the major cause of hyperglycemic oxidative stress in this tissue. The substantial decrease in the level of reduced glutathione (GSH) with concomitant rise in the level of lipid peroxidn. product malondialdehyde (MDA) in the lens of transgenic mice, but not in the nontransgenic mice, suggests that glucose autoxidn. and nonenzymic glycation do not contribute to oxidative stress in diabetic lenses. AR redn. of glucose to sorbitol probably contributes to oxidative stress by depleting its cofactor NADPH, which is also required for the regeneration of GSH. Sorbitol dehydrogenase, the 2nd enzyme in the polyol pathway that converts sorbitol to fructose, also contributes to oxidative stress, most likely because depletion of its cofactor NAD+ leads to more glucose being channeled through the polyol pathway. Despite a >100% increase of MDA, oxidative stress plays only a minor role in the development of cataract in this acute diabetic cataract model. Chronic oxidative stress generated by the polyol pathway is likely to be an important contributing factor in the slow-developing diabetic cataract as well as in the development of other diabetic complications.

REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 12 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 1998:618371 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 129:255004  
TITLE: Prophylactic and therapeutic methods for ocular degenerative diseases and inflammations, and histidine compositions therefor  
INVENTOR(S): Thomas, Peter G.  
PATENT ASSIGNEE(S): Cytos Pharmaceuticals LLC, USA  
SOURCE: U.S., 10 pp.  
CODEN: USXXAM  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

| PATENT NO.                                                                                                                                                                                                                                                                                                                    | KIND | DATE     | APPLICATION NO. | DATE       |
|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------|----------|-----------------|------------|
| US 5811446                                                                                                                                                                                                                                                                                                                    | A    | 19980922 | US 1997-839805  | 19970418   |
| WO 9847366                                                                                                                                                                                                                                                                                                                    | A1   | 19981029 | WO 1998-US7319  | 19980417   |
| W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM |      |          |                 |            |
| RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG                                                                                                                                                        |      |          |                 |            |
| AU 9873583                                                                                                                                                                                                                                                                                                                    | A1   | 19981113 | AU 1998-73583   | 19980417   |
| PRIORITY APPLN. INFO.:                                                                                                                                                                                                                                                                                                        |      |          | US 1997-839805  | A 19970418 |
|                                                                                                                                                                                                                                                                                                                               |      |          | WO 1998-US7319  | W 19980417 |

AB Methods are provided for protecting the eye from degenerative eye conditions by administering prophylactic histidine compns. Also provided are for treating ocular inflammation resulting from various causative agents, by administering therapeutic histidine compns. Further provided are histidine compns. for carrying out the methods.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 13 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 1998:608534 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 129:225723  
TITLE: The use of heme-peptides to prevent or retard disease

INVENTOR(S): associated with oxidative stress  
 Spector, Abraham; Ma, Wanchao; Wang, Ren-Rong  
 PATENT ASSIGNEE(S): The Trustees of Columbia University in the City of New  
 York, USA  
 SOURCE: PCT Int. Appl., 74 pp.  
 CODEN: PIXXD2  
 DOCUMENT TYPE: Patent  
 LANGUAGE: English  
 FAMILY ACC. NUM. COUNT: 1  
 PATENT INFORMATION:

| PATENT NO.                                                             | KIND | DATE     | APPLICATION NO. | DATE        |
|------------------------------------------------------------------------|------|----------|-----------------|-------------|
| WO 9837908                                                             | A1   | 19980903 | WO 1998-US3857  | 19980227    |
| W: AU, CA, JP, MX, US                                                  |      |          |                 |             |
| RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE |      |          |                 |             |
| US 6013623                                                             | A    | 20000111 | US 1997-807482  | 19970227    |
| AU 9864424                                                             | A1   | 19980918 | AU 1998-64424   | 19980227    |
| PRIORITY APPLN. INFO.:                                                 |      |          | US 1997-807482  | A2 19970227 |
|                                                                        |      |          | WO 1998-US3857  | W 19980227  |

AB A method for treating a condition assocd. with oxidative stress in a  
 subject comprises administering an effective amt. of a heme-peptide. The  
 subject may be a mammal. The mammal may be a human being. The condition  
 assocd. with oxidative stress may be an inflammatory condition, an  
 allergic condition or an auto-immune condition.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS  
 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 14 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1998:206249 CAPLUS <<LOGINID::20060721>>

DOCUMENT NUMBER: 128:280319

TITLE: UVA irradiation of human lens proteins produces  
 residual oxidation of ascorbic acid even in the  
 presence of high levels of glutathione

AUTHOR(S): Ortwerth, Beryl J.; Coots, Amy; James, Hongying L.;  
 Linetsky, Mikhail

CORPORATE SOURCE: Mason Eye Institute, University of Missouri, Columbia,  
 MO, 65212, USA

SOURCE: Archives of Biochemistry and Biophysics (1998),  
 351(2), 189-196  
 CODEN: ABBIA4; ISSN: 0003-9861

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The oxidn. products of ascorbic acid (AsCH-) can rapidly glycate and  
 crosslink lens proteins in \*\*\*vitro\*\*\*, producing fluorophores and  
 browning products similar to those present in cataractous lenses. The  
 accumulation of AsCH- oxidn. products, however, would largely be prevented  
 by the millimolar levels of glutathione (GSH) present in human lens. Here  
 we investigate whether protein aggregation could allow the oxidn. of AsCH-  
 by UVA-induced reactive oxygen species in the presence of physiol. levels  
 of GSH. The metal-catalyzed oxidn. of 1.0 mM AsCH- by 50 .mu.M Cu(II) was  
 almost complete after 1 h, but no oxidn. was seen in the presence of GSH  
 concns. as low as 0.5 mM. UVA irradiation of protein aggregates from human  
 lens, which accumulated more than 2.0 mM singlet oxygen after 1 h, caused  
 a 50-60% oxidn. of 1.0 mM AsCH-. The addn. of 2-4 mM GSH, however,  
 decreased AsCH- oxidn. by less than half, and 30% of the AsCH- was  
 oxidized even in the presence of 15 mM GSH. This diminished protection  
 may be due, in part, to the ability of AsCH-, but not GSH, to penetrate to  
 the sites of singlet oxygen generation located within the protein.  
 Consistent with this hypothesis, greater GSH protection was seen when a  
 proteolytic digest of the human proteins was subjected to the same irradiation.  
 or when singlet oxygen was chem. generated from 3-(4-methyl-1-  
 naphthyl)propionic acid endoperoxide (MNPAE) at 37.degree.C in the medium.  
 The addn. of 50 .mu.M Cu(II) had no effect on the rate of degrdn. of  
 dehydroascorbic acid (DHA). Singlet oxygen, either UVA- or  
 MNPAE-generated, increased the rate of DHA loss. This secondary oxidn. of  
 DHA by singlet oxygen would allow the accumulation of AsCH- oxidn.  
 products not reducible by GSH. Therefore, the data presented here argue  
 that the protein aggregation seen in older human lenses may permit  
 oxidized AsCH--induced crosslinking to occur even at physiol. GSH levels.

REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS



L20 ANSWER 15 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN  
 ACCESSION NUMBER: 1997:608376 CAPLUS <<LOGINID::20060721>>  
 DOCUMENT NUMBER: 127:272771  
 TITLE: Progression of mouse buthionine sulfoximine  
 \*\*\*cataracts\*\*\* in \*\*\*vitro\*\*\* is inhibited by  
 thiols or ascorbate  
 AUTHOR(S): Calvin, Harold I.; Zhu, Guan-Ping; Wu, Jun-Xi;  
 Banerjee, Urmil; Fu, S. -C. Joseph  
 CORPORATE SOURCE: Department of Ophthalmology and Department of  
 Biochemistry and Molecular Biology, UMDNJ-New Jersey  
 Medical School, Newark, NJ, 07103, USA  
 SOURCE: Experimental Eye Research (1997), 65(3), 341-347  
 CODEN: EXERA6; ISSN: 0014-4835  
 PUBLISHER: Academic  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English

AB Mouse lens cultures were employed to study the progression of cataracts initiated by injection of buthionine sulfoximine, an inhibitor of glutathione (GSH) biosynthesis. Culture of lenses removed from untreated mice on postnatal day 7, for 48 h in the presence of 4 mM BSO, resulted in only limited cataractous changes. To enable substantial progression of cataracts in \*\*\*vitro\*\*\*, it was therefore necessary to pretreat the mice with BSO prior to lens culture. A single injection of BSO (4 nmol mg<sup>-1</sup> lens), administered on day 7, resulted in > 90% depletion of lens GSH within 3 days, but no visible cataractous changes. The clear lenses were incubated for 29 h at 37.degree. in Medium HL-1, supplemented with EGF, insulin and Ca<sup>2+</sup>, in the presence or absence of BSO, and were scored for cataract development by previously described criteria. In the absence of BSO, only 4 of 10 lenses developed large opacities. However, in the presence of 4 mM BSO, 40 out of 45 exptl. lenses developed opacities affecting at least 50% of the lens visual field and were scored as stages 1C-4, depending upon the extent and d. of the cataracts. In addn., three lenses had opacities involving 20-50% of the field (stage 1B). By contrast, less than 10% of lenses from untreated mice incubated in the absence of BSO developed opacities. The cataracts developed in 4 mM BSO were accompanied by redn. of lens glutathione levels to < 0.010 nmol mg<sup>-1</sup> lens. They were almost completely prevented by 1 mM ascorbate, 2 mM GSH, 2 mM GSH monoethyl ester and 2 mM cysteamine. GSH and GSH ester maintained lens glutathione content between 0.1 and 0.2 nmol mg<sup>-1</sup> in the presence of BSO, whereas ascorbate did not prevent near-total GSH depletion. The prevention of cataracts by thiols and ascorbate was confirmed by lens Na/K ratios not significantly different from those in control lenses. The above combination of GSH depletion in vivo by a single injection of BSO, followed 3 days later with lens culture in the presence of BSO, may yield a useful system to elucidate and control the biochem. mechanisms involved in oxidative cataract induction by this GSH-depleting agent.

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> file his  
 'HIS' IS NOT A VALID FILE NAME  
 SESSION CONTINUES IN FILE 'CAPLUS'  
 Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files that are available. If you have requested multiple files, you can specify a corrected file name or you can enter "IGNORE" to continue accessing the remaining file names entered.

=> d his

(FILE 'HOME' ENTERED AT 15:13:55 ON 21 JUL 2006)

FILE 'CAPLUS' ENTERED AT 15:14:19 ON 21 JUL 2006

FILE 'REGISTRY' ENTERED AT 15:14:41 ON 21 JUL 2006  
 E REDUCED GLUTATHIONE/CN

FILE 'CAPLUS' ENTERED AT 15:14:41 ON 21 JUL 2006  
 S E3

L1 FILE 'REGISTRY' ENTERED AT 15:14:47 ON 21 JUL 2006  
1 S E3/CN

L2 FILE 'CAPLUS' ENTERED AT 15:14:48 ON 21 JUL 2006  
41636 S L1  
L3 1 S 70-18-8/THU AND DILLON J?/AU  
L4 1 S US20040229814/PN  
SELECT L4 1 RN

L5 FILE 'REGISTRY' ENTERED AT 15:17:38 ON 21 JUL 2006  
4 S E1-E4  
L6 1 S 50-81-7/RN  
L7 1 S 7727-37-9/RN  
L8 1 S 7782-44-7/RN  
L9 0 S (VITREOUS(A)REPLACEMENT) AND 70-18-8 AND 50-81-7  
L10 0 S (VITREOUS(A)REPLACEMENT) AND 70-18-8/RN

L11 FILE 'CAPLUS' ENTERED AT 15:27:46 ON 21 JUL 2006  
E VITREOUS REPLACEMENT/CT  
6087 S CATARACT/OBI  
S 70-18-8/REG# AND 50-81-7/REG#

L12 FILE 'REGISTRY' ENTERED AT 15:29:30 ON 21 JUL 2006  
1 S 50-81-7/RN

L13 FILE 'CAPLUS' ENTERED AT 15:29:31 ON 21 JUL 2006  
78897 S L12

L14 FILE 'REGISTRY' ENTERED AT 15:29:31 ON 21 JUL 2006  
1 S 70-18-8/RN

L15 FILE 'CAPLUS' ENTERED AT 15:29:32 ON 21 JUL 2006  
41636 S L14  
L16 4494 S L15 AND L13  
L17 77 S L16 AND L11  
L18 75 S L17 NOT DILLON J?/AU  
L19 64 S L18 NOT PY>2002  
L20 36 S L19 AND VIT?

=> s l20 and vitrectom?

L21 0 L20 AND VITRECTOM?

=> d l20 15-30 ibib abs

L20 ANSWER 15 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 1997:608376 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 127:272771  
TITLE: Progression of mouse buthionine sulfoximine  
\*\*\*cataracts\*\*\* in \*\*\*vitro\*\*\* is inhibited by  
thiols or ascorbate  
AUTHOR(S): Calvin, Harold I.; Zhu, Guan-Ping; Wu, Jun-Xi;  
Banerjee, Urmi; Fu, S. -C. Joseph  
CORPORATE SOURCE: Department of Ophthalmology and Department of  
Biochemistry and Molecular Biology, UMDNJ-New Jersey  
Medical School, Newark, NJ, 07103, USA  
SOURCE: Experimental Eye Research (1997), 65(3), 341-347  
CODEN: EXERA6; ISSN: 0014-4835  
PUBLISHER: Academic  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Mouse lens cultures were employed to study the progression of cataracts initiated by injection of buthionine sulfoximine, an inhibitor of glutathione (GSH) biosynthesis. Culture of lenses removed from untreated mice on postnatal day 7, for 48 h in the presence of 4 mM BSO, resulted in only limited cataractous changes. To enable substantial progression of cataracts in \*\*\*vitro\*\*\*, it was therefore necessary to pretreat the mice with BSO prior to lens culture. A single injection of BSO (4 nmol mg-1 lens), administered on day 7, resulted in > 90% depletion of lens GSH within 3 days, but no visible cataractous changes. The clear lenses were incubated for 29 h at 37.degree. in Medium HL-1, supplemented with EGF, insulin and Ca2+, in the presence or absence of BSO, and were scored for

cataract development by previously described criteria. In the absence of BSO, only 4 of 10 lenses developed large opacities. However, in the presence of 4 mM BSO, 40 out of 45 exptl. lenses developed opacities affecting at least 50% of the lens visual field and were scored as stages 1C-4, depending upon the extent and d. of the cataracts. In addn., three lenses had opacities involving 20-50% of the field (stage 1B). By contrast, less than 10% of lenses from untreated mice incubated in the absence of BSO developed opacities. The cataracts developed in 4 mM BSO were accompanied by redn. of lens glutathione levels to < 0.010 nmol mg<sup>-1</sup> lens. They were almost completely prevented by 1 mM ascorbate, 2 mM GSH, 2 mM GSH monoethyl ester and 2 mM cysteamine. GSH and GSH ester maintained lens glutathione content between 0.1 and 0.2 nmol mg<sup>-1</sup> in the presence of BSO, whereas ascorbate did not prevent near-total GSH depletion. The prevention of cataracts by thiols and ascorbate was confirmed by lens Na/K ratios not significantly different from those in control lenses. The above combination of GSH depletion in vivo by a single injection of BSO, followed 3 days later with lens culture in the presence of BSO, may yield a useful system to elucidate and control the biochem. mechanisms involved in oxidative cataract induction by this GSH-depleting agent.

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 16 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1996:242279 CAPLUS <<LOGINID::20060721>>

DOCUMENT NUMBER: 124:307535

TITLE: Stereospecific effects of R-lipoic acid on buthionine sulfoximine-induced \*\*\*cataract\*\*\* formation in newborn rats

AUTHOR(S): Maitra, Indrani; Serbinova, Elena; Tritscheler, Hans; Packer, Lester

CORPORATE SOURCE: Dep. Molecular and Cell Biol., Univ. California, Berkeley, CA, 94720-3200, USA

SOURCE: Biochemical and Biophysical Research Communications (1996), 221(2), 422-9  
CODEN: BBRCA9; ISSN: 0006-291X

PUBLISHER: Academic

DOCUMENT TYPE: Journal

LANGUAGE: English

AB This study revealed a marked stereospecificity in the prevention of buthionine sulfoximine-induced cataract, and in the protection of lens antioxidants, in newborn rats by .alpha.-lipoate. R- and racemic .alpha.-lipoate decreased cataract formation from 100% (buthionine sulfoximine only) to 55% (buthionine sulfoximine + R-.alpha.-lipoic acid) and 40% (buthionine sulfoximine + rac-.alpha.-lipoic acid) (p < 0.05 compared to buthionine sulfoximine only). S-.alpha.-lipoic acid had no effect on cataract formation induced by buthionine sulfoximine. The lens antioxidants glutathione, ascorbate, and \*\*\*vitamin\*\*\* E were depleted to 45, 62, and 23% of control levels, resp., by buthionine sulfoximine treatment, but were maintained at 84-97% of control levels when R-.alpha.-lipoic acid or rac-.alpha.-lipoic acid were administered with buthionine sulfoximine; S-.alpha.-lipoic acid administration had no protective effect on lens antioxidants. When enantiomers of .alpha.-lipoic acid were administered to animals, R-.alpha.-lipoic acid was taken up by lens and reached concns. 2- to 7-fold greater than those of S-.alpha.-lipoic acid, with rac-.alpha.-lipoic acid reaching levels midway between the R-isomer and racemic form. Reduced lipoic acid, dihydrolipoic acid, reached the highest levels in lens of the rac-.alpha.-lipoic acid-treated animals and the lowest levels in S-.alpha.-lipoic acid-treated animals. These results indicate that the protective effects of .alpha.-lipoic acid against buthionine sulfoximine-induced cataract are probably due to its protective effects on lens antioxidants, and that the stereospecificity exhibited is due to selective uptake and redn. of R-.alpha.-lipoic acid by lens cells.

L20 ANSWER 17 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1996:83797 CAPLUS <<LOGINID::20060721>>

DOCUMENT NUMBER: 124:194229

TITLE: Effect of selected anti- \*\*\*cataract\*\*\* agents on opacification in the selenite \*\*\*cataract\*\*\* model

AUTHOR(S): Hiraoka, T.; Clark, J. I.; Li, X. Y.; Thurston, G. M.

CORPORATE SOURCE: Dep. Biol. Structure, Univ. Washington, Seattle, WA,

98195, USA

SOURCE: Experimental Eye Research (1996), 62(1), 11-19  
CODEN: EXERA6; ISSN: 0014-4835  
PUBLISHER: Academic  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB A systematic study of the anti-cataract activity of 14 reagents was conducted using the selenite model. The reagents or their derivs. were identified from literature reports of their potential effectiveness against cataract formation. The effects of each reagent were measured on the phase sepn. temp., Tc, of lens homogenate in \*\*\*vitro\*\*\*. Tc is a direct measure of mol. interaction leading to protein aggregation. The protective effect of a single s.c. injection of each reagent [at a dose of 1.5 mmol/kg body wt.] on lens opacification was evaluated in vivo using rats administered selenite [at a dose of 19 .mu.mol/kg body wt.] to initiate cataract formation. The strongest effects on lens opacification in vivo were obsd. with reagents having the strongest effect on Tc, in \*\*\*vitro\*\*\*. The weakest effects in vivo were obsd. with the reagents having the weakest effect on Tc, in \*\*\*vitro\*\*\*. The results were suggestive of a relation between the effect of reagent on Tc and protection against cataract formation in vivo.

L20 ANSWER 18 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1995:932639 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 124:49578  
TITLE: Morphological and biochemical changes in lenses of guinea pigs after \*\*\*vitamin\*\*\* -C-deficient diet and UV-B radiation  
AUTHOR(S): Malik, A.; Kojima, M.; Sasaki, K.  
CORPORATE SOURCE: Faculty Medicine, University Andalas, Padang, Indonesia  
SOURCE: Ophthalmic Research (1995), 27(4), 189-96  
CODEN: OPRSAQ; ISSN: 0030-3747  
PUBLISHER: Karger  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB The effect of UV B (UV-B) radiation and a \*\*\*vitamin\*\*\* -C-deficient (VCD) diet on guinea pig lenses was investigated. The initial lens changes in the VCD group were obsd. by slit-lamp examn. 6 wk after the start of the VCD treatment; after 12 wk the changes in the posterior subcapsular region became more prominent, and the dissocn. around the posterior suture became wider and slightly deeper toward the posterior cortex. The high concn. of lens oxidized glutathione (GSSG), and the low ratio of reduced glutathione (GSH) to oxidized glutathione (GSH/GSSG) on the lens posterior region correlated with d. changes in the corresponding layers as measured by Scheimpflug images with linear microdensitometry. It is suggested that the strong oxidative stress of the VCD diet caused the damage at the posterior part of the lens. UV-B radiation appeared to accelerate cataract progression in lenses that lack \*\*\*vitamin\*\*\* C.

L20 ANSWER 19 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1995:771303 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 123:246741  
TITLE: Biochemical changes in lens, aqueous humor and \*\*\*vitreous\*\*\* body and effects of aldose reductase inhibitor (TAT) on rats with experimental diabetes  
AUTHOR(S): Saito, Hitoko  
CORPORATE SOURCE: Dep. Ophthalmol., Nippon Med. Sch., Tokyo, 113, Japan  
SOURCE: Nippon Ika Daigaku Zasshi (1995), 62(4), 339-50  
CODEN: NIDZAJ; ISSN: 0048-0444  
DOCUMENT TYPE: Journal  
LANGUAGE: Japanese

AB We measured the levels of antioxidants (glutathione, ascorbic acid) and lipid peroxide (malondialdehyde) in lens, aq. humor and \*\*\*vitreous\*\*\* body of rats with galactose cataract and streptozotocin cataract. Furthermore we studied the effects of aldose reductase inhibitor (TAT) on these levels. In streptozotocin diabetes rats, the increased malondialdehyde levels in lens, aq. humor and serum were suppressed by TAT administration. In galactose and streptozotocin diabetes rats, the decreased levels of glutathione and ascorbic acid were suppressed by TAT administration.

L20 ANSWER 20 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN  
 ACCESSION NUMBER: 1995:372990 CAPLUS <<LOGINID::20060721>>  
 DOCUMENT NUMBER: 122:211148  
 TITLE: Participation of oxidative damage in cataractogenesis in WBN/Kob rats with spontaneously, late developed diabetes mellitus  
 AUTHOR(S): Goto, Hajime; Okada, Hiroshi; Hattori, Hiroyuki; Majima, Yoshinao; Ohta, Yoshiji; Ishiguro, Isao  
 CORPORATE SOURCE: Sch. Med., Fujita Health Univ., Toyoake, 470-11, Japan  
 SOURCE: Atarashii Ganka (1995), 12(1), 103-8  
 CODEN: ATGAEX; ISSN: 0910-1810  
 DOCUMENT TYPE: Journal  
 LANGUAGE: Japanese  
 AB We examd. the relation between severity of cataract and lenticular glycated protein, lipid peroxide (LPO), \*\*\*vitamin\*\*\* E (VE), reduced glutathione (GSH), and ascorbic acid (AA) levels, as well as superoxide dismutase (SOD) activity, in male diabetic WBN/Kob rats (22-mo-old). In the lenses of diabetic rats without cataract, increased glycated protein and LPO contents and decreased AA content were found. In the lenses of diabetic rats with cataract, increased VE content and decreased GSH content and SOD activity occurred in addn. to the increased glycated protein and LPO contents and decreased AA content. These changes were enhanced in diabetic rats with advanced cataract. In diabetic rats with and without cataract, the levels of blood sugar, serum insulin, and glycated protein were similar. In the serum of these diabetic rats, increased LPO and VE levels and decreased AA level were almost equal. These results indicate that oxidative damage participates in cataractogenesis in WBN/Kob rats with spontaneously, late developed diabetes mellitus.

L20 ANSWER 21 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN  
 ACCESSION NUMBER: 1995:219383 CAPLUS <<LOGINID::20060721>>  
 DOCUMENT NUMBER: 122:53130  
 TITLE: Relationship between cataractogenesis and oxidative damage in WBN/Kob rats with spontaneously late-developed diabetes mellitus  
 AUTHOR(S): Goto, Hajime; Okada, Hiroshi; Hattori, Hiroyuki; Majima, Yoshinao; Ohta, Yoshiji; Ishiguro, Isao  
 CORPORATE SOURCE: Sch. Med., Fujita Health Univ., Toyoake, 470-11, Japan  
 SOURCE: Atarashii Ganka (1994), 11(10), 1599-603  
 CODEN: ATGAEX; ISSN: 0910-1810  
 DOCUMENT TYPE: Journal  
 LANGUAGE: Japanese  
 AB To clarify the role of oxidative damage in cataractogenesis in diabetic persons of middle or advanced age, we examd. the relationship between cataractogenesis and oxidative damage in WBN/Kob rats with spontaneously late developed diabetes mellitus. Male WBN/Kob rats (17 mo old), with diabetes and cortical cataract, and age-matched male Wistar rats, without diabetes or cataract, were used as exptl. and control groups, resp. Cataractous lenses in the exptl. group contained 2.5 fold the amt. of glycated protein found in the clear lenses of the control group. Lipid peroxide content in the cataractous lenses of the exptl. group was significantly higher than in the clear lenses of the control group, while reduced glutathione, ascorbic acid, and \*\*\*vitamin\*\*\* E contents in the former were significantly lower than in the latter. These results suggest a close relationship between cataractogenesis and oxidative damage in WBN/Kob rats with spontaneously late developed diabetes mellitus.

L20 ANSWER 22 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN  
 ACCESSION NUMBER: 1993:37067 CAPLUS <<LOGINID::20060721>>  
 DOCUMENT NUMBER: 118:37067  
 TITLE: The analysis of \*\*\*vitamin\*\*\* C, glutathione, and lipid peroxides in senile \*\*\*cataract\*\*\* lens  
 AUTHOR(S): Wu, Xiangyun; Hu, Xiaoyan; Li, Han; Xu, Songde  
 CORPORATE SOURCE: Dep. Biochem., Shandong Med. Univ., Jinan, Peop. Rep. China  
 SOURCE: Shandong Yike Daxue Xuebao (1992), 30(2), 147-8  
 CODEN: SYXBEE; ISSN: 1000-0496  
 DOCUMENT TYPE: Journal  
 LANGUAGE: Chinese  
 AB The level of \*\*\*vitamin\*\*\* C, GSH, and lipid peroxide in 20 cases of senile cataract lens and 5 cases of noncataract lens were analyzed. The

levels of \*\*\*vitamin\*\*\* C and GSH were decreased and the level of lipid peroxides was increased in the senile cataract lens. The effect of the oxidant and antioxidant might be involved in the formation of senile cataract. It suggested that there may be some relationship between the content of \*\*\*vitamin\*\*\* C, G-SH and lipid peroxides of the lens and the pathogenesis of the senile cataract.

L20 ANSWER 23 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 1992:91439 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 116:91439  
TITLE: Composition and method for treatment of macular degeneration  
INVENTOR(S): Lahaye, Peter G.; Olson, Randall J.  
PATENT ASSIGNEE(S): Lahaye Laboratories, Inc., USA  
SOURCE: U.S., 6 pp.  
CODEN: USXXAM  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
FAMILY ACC. NUM. COUNT: 1  
PATENT INFORMATION:

| PATENT NO.             | KIND | DATE     | APPLICATION NO. | DATE        |
|------------------------|------|----------|-----------------|-------------|
| US 5075116             | A    | 19911224 | US 1989-341025  | 19890420    |
| US 5156852             | A    | 19921020 | US 1991-761694  | 19910918    |
| PRIORITY APPLN. INFO.: |      |          | US 1989-341025  | A2 19890420 |

AB Oral pharmaceutical compns. for treatment of eye diseases such as macular degeneration are disclosed. The compns. contain \*\*\*vitamin\*\*\* C and E, Zn, Cu, Se, Mn, and at least one of L-cysteine, pyridoxine, and riboflavin. The \*\*\*vitamins\*\*\* C and E serve as antioxidants, while Zn, Cu, Se, and Mn serve as cofactors for metalloenzymes which scavenge oxidizers. The remaining three compds. tend to enhance glutathione concn. All the elements are provided in a tablet and taken 4 tablets twice a day (no data).

L20 ANSWER 24 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 1991:464712 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 115:64712  
TITLE: Desferal-manganese(III) in the therapy of diquat-induced \*\*\*cataract\*\*\* in rabbit  
AUTHOR(S): Bhuyan, Kailash C.; Bhuyan, Durga K.; Chiu, William; Malik, Sajid; Fridovich, Irwin  
CORPORATE SOURCE: Dep. Ophthalmol., Mt. Sinai Sch. Med., New York, NY, 10029, USA  
SOURCE: Archives of Biochemistry and Biophysics (1991), 288(2), 525-32  
CODEN: ABBIA4; ISSN: 0003-9861  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB In rabbit eye lenses subjected to oxidative stress induced by 1 mM diquat in \*\*\*vitro\*\*\*, there were 7-10-fold increases in malondialdehyde, conjugated dienes, and carbonyl dienes, indicating extensive peroxidn. of cellular membrane lipids, and .apprx.60% decrease in reduced glutathione. In the presence of 0.1-5 mM desferal-Mn(III) these changes were diminished by 50-70%. In rabbits having diquat-induced cataract, 5% desferal-Mn(III) applied topically four times a day and a single i.p. dose of 64 mg/kg daily for 5 wk (including pretreatment for 1 wk) retarded the progression of lens opacities, whereas in control rabbits cataract progressed to an advanced grade. Desferal-Mn(III) also diminished prodn. of superoxide and hydroxyl radicals in the lens, aq. humor, and \*\*\*vitreous\*\*\* humor, and of H2O2 in the aq. humor and \*\*\*vitreous\*\*\* humor. It also suppressed lipid peroxidn. and oxidn. of protein-SH of the lens and restored lenticular glutathione and ascorbate to normal levels.

L20 ANSWER 25 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN  
ACCESSION NUMBER: 1990:5633 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 112:5633  
TITLE: Chromium-51 release and oxidative stress in the lens  
AUTHOR(S): Stewart-DeHaan, P. J.; Sanwal, M.; Creighton, M. Q.; Inch, W. R.; Trevithick, J. R.  
CORPORATE SOURCE: Dep. Biochem., Univ. West. Ontario, London, ON, N6A 5C1, Can.

SOURCE: Lens and Eye Toxicity Research (1989), 6(1-2), 183-202  
CODEN: LETRET; ISSN: 1042-6922  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Examn. of the opaque areas of human cortical cataracts has shown that a large portion of the opacity could be attributed to the globules found there. Models were tested which featured globule formation as a result of oxidative damage to cultured rat lens cells and whole chick embryo lenses. When cell monolayers from a lens cell line were exposed to oxidizing conditions they developed globules on the cell surface. The cells were protected from damage by the addn. of glutathione and \*\*\*vitamin\*\*\* C. Lenses of 13-day-old chick embryos were also incubated in oxidizing conditions and the amt. of cellular damage was assessed using a chromium-51 release assay. After 24 h the amt. of 51Cr in the medium increased by 20% as a result of 10 mM hydrogen peroxide treatment. The addn. of 10 mM \*\*\*vitamin\*\*\* C to the hydrogen peroxide reduced the 51Cr leakage to the control level. Light microscopy of sections of the lens showed a breakdown of the equatorial fiber arrangement in the presence of H2O2, while addn. of \*\*\*vitamin\*\*\* C restored the fiber organization to almost normal. Oxidative stress may be an important step in cataractogenesis and water-sol. antioxidants may be protective agents.

L20 ANSWER 26 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1989:587544 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 111:187544  
TITLE: Effects of antioxidants or free radical scavengers on  
\*\*\*cataract\*\*\* formation induced by selenium  
AUTHOR(S): Huang, Lili; Jia, Weihong; Chang, Changying  
CORPORATE SOURCE: Dep. Biochem., Beijing Med. Univ., Beijing, Peop. Rep. China  
SOURCE: Shengwu Huaxue Zazhi (1989), 5(4), 369-74  
CODEN: SHZAE4; ISSN: 1000-8543  
DOCUMENT TYPE: Journal  
LANGUAGE: Chinese

AB The antioxidants or free radical scavengers GSH, \*\*\*vitamin\*\*\* E, \*\*\*vitamin\*\*\* C, mannitol, DMSO, and 3 synthetic anticataract compds. (AC1, AC2, and AC3) inhibited cataract formation induced by Se (Na2SeO3) in rats.

L20 ANSWER 27 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1989:491894 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 111:91894  
TITLE: In \*\*\*vitro\*\*\* studies on the effect of cadmium on goat eye lens  
AUTHOR(S): Srivastava, V. K.; Pandey, D. C.; Varshney, N.  
CORPORATE SOURCE: Dep. Chem., Univ. Gorakhpur, Gorakhpur, 273 009, India  
SOURCE: Current Science (1989), 58(12), 712-13  
CODEN: CUSCAM; ISSN: 0011-3891  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB Lenses were isolated from goat eyes and incubated with or without Cd and the lens levels of ascorbic acid, GSH, and proteins detd.; in the presence of Cd, a whitish coating developed on the lenses. Concns. of GSH and ascorbic acid decreased in lenses exposed to Cd in a concn. dependent manner. Sol. proteins also decreased and insol. proteins increased. The relations of the obsd. changes to cataract development during Cd exposure was discussed.

L20 ANSWER 28 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1989:55353 CAPLUS <<LOGINID::20060721>>  
DOCUMENT NUMBER: 110:55353  
TITLE: Change of ascorbic acid in rat lenses in the course of their \*\*\*cataract\*\*\* development initiated by galactose  
AUTHOR(S): Lee, Teguk; Iwamoto, Takeo; Hitomi, Elza; Kanematsu, Takata; Yoshiura, Masahiko; Oinuma, Shinichi; Iriyama, Keiji  
CORPORATE SOURCE: Sch. Med., Teikyo Univ., Japan  
SOURCE: Jikeikai Medical Journal (1988), 35(3), 225-37  
CODEN: JMEJAS; ISSN: 0021-6968  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB A change in ascorbic acid, a water-sol. antioxidant, in rat lenses during the course of galactose-induced opacification was re-examined by a selective, sensitive, and reliable high-performance liq. chromatog.-electrochem. detection method. Compared to the control group, the easily oxidizable water-sol. \*\*\*vitamin\*\*\* decreased in the lenses of the galactose-fed group with the development of opacification. The plausible role of glutathione and uric acid as the potent water-sol. antioxidants is discussed and compared with that of ascorbic acid.

L20 ANSWER 29 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1987:590 CAPLUS <<LOGINID::20060721>>

DOCUMENT NUMBER: 106:590

TITLE: Antioxidants in prevention of oxidative damage to the lens and \*\*\*cataract\*\*\* in vivo

AUTHOR (S) : Bhuyan, Durga K.; Podos, Steven M.; Bhuyan, Kailash C.

CORPORATE SOURCE: Mt. Sinai Sch. Med., City Univ. New York, New York,  
NY, 10029, USA

SOURCE: Superoxide Superoxide Dismutase Chem., Biol. Med., Proc. Int. Conf., 4th (1986), Meeting Date 1985, 657-61. Editor(s): Rotilio, Giuseppe. Elsevier: Amsterdam. Neth.

CODEN: 55GJAL

DOCUMENT TYPE: Conference: General Review

LANGUAGE: English

AB The therapy of cataracts with antioxidants was studied in animal models of cataract (3-aminotriazole-induced and galactose-induced in rabbits and selenium-induced in rats). \*\*\*Vitamin\*\*\* E [1406-18-4] was effective in preventing both rabbit models of cataract and centrophenoxine [3685-84-5] was effective in arresting the progression of the galactose cataract model. In addn., these compds. decreased lens damage (lipid peroxidn.) in these models of cataract. The protective effects of antioxidants on oxidative stress-induced damage (lipid peroxidn.) were also studied in rabbit lens in \*\*\*vitro\*\*\*.

L20 ANSWER 30 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 1986:532646 CAPLUS <<LOGINID::20060721>>

DOCUMENT NUMBER: 105:132646

TITLE: Prevention of diabetic cataractogenesis in streptozotocin-treated rats by subsequent \*\*\*vitamin\*\*\* E administration

AUTHOR(S) : Hirai, Tatsuo; Majima, Yoshinao; Nakamura, Kimi; Ohta, Yoshiji; Ishiguro, Isao

CORPORATE SOURCE: Sch. Med., Fujita-Gakuen Health Univ., Toyoake, Japan

SOURCE: Atarashii Ganka (1986), 3(2), 247-50

CODEN: ATGAEX; ISSN: 0910-1810

DOCUMENT TYPE: Journal

LANGUAGE : Japanese

AB d-.alpha.-Tocopherol [59-02-9] was given to rats at 335 mg/kg-day i.m. for 10 days, beginning 5 days after a single injection of streptozotocin (50 mg/kg, i.p.). Tocopherol administration inhibited lenticular vacuole formation and the loss of tocopherol assocd. with diabetogenic cataracts. It also reduced lenticular lipid accumulation but did not affect the diabetes-related decreases of GSH [ \*\*\*70-18-8\*\*\* ] and ascorbic acid [ \*\*\*50-81-7\*\*\* ] or the accumulation of sorbitol [50-70-4] in the lens. Lenticular \*\*\*vitamin\*\*\* E is considered to play a crit. role in diabetic cataract formation.

$$\Rightarrow \log y$$

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

## SESSION

FULL ESTIMATED COST

114.13

169.44

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE

TOTAL

## ENTRY

## SESSION

CA SUBSCRIBER PRICE

-23.25

-23-25

STN INTERNATIONAL LOGOFF AT 15:49:03 ON 21 JUL 2006